Abstract presentado en el XVth Congress of the European Haematology Association, a celebrar en Barcelona del 10-13 Junio de 2010

MUTATIONAL ANALYSIS OF THE EGF RECEPTOR GENE IN BCR-ABL1 NEGATIVE AND JAK2V617F NEGATIVE CHRONIC MYELOPROLIFERATIVE NEOPLASMS

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Background: BCR-ABL1 negative chronic myeloproliferative neoplasms (CMPNs) are a heterogeneous group of clonal hematological malignancies. In last years, some genetic alterations have been described to cause these diseases, most of them activating tyrosine kinase (TK) genes. Tyrosine kinases proteins have an important role in cell growth and oncogenesis. Gain-of-function mutations in TK genes can produce a constitutive activation of several signaling pathways.

Aims: In this study, we study the EGFR gene that codes for a tyrosine kinase receptor (RTK) involved in signaling pathways relevant in hematological cells. Mutations in this gene have been found involved in lung cancer, and it could have an important role in the pathogenesis of hematological disorders.

Methods: We have analyzed the transmembrane and TK-coding domains of EGFR by dHPLC to detect mutations on samples from 44 BCR-ABL1 negative / V617FJAK2 negative CMPN patients and 20 control samples from healthy individuals.

Results: Our results show that this gene is not frequently mutated in CMPNs.

Conclusion: The EGF Receptor gene does not appear to be involved in the pathogenesis of the myeloproliferative neoplasms.

This work has been funded with the help of the Institute of Health Carlos III (FIS PI040037), Spanish Ministry of Science and Innovation (SAF 2007-62473), the PIUNA Program of the University of Navarra and the Caja Navarra Foundation through the Program “You choose, you decide” (Project 10830).